## Amendments to the Claims

Claim 1. (Currently Amended) A method of detecting genetic predisposition to venous thrombosis (VT) in a <u>human</u> subject, comprising:

determining whether screening the human subject has one or more mutations or polymorphisms in at least 143 of the for the presence of the 143 mutations or polymorphisms listed in Table 1, wherein the mutations or polymorphisms are present in at least eight VT-related molecules comprising antithrombin III (AT III), protein C, protein S, fibrinogen, factor V (FV), prothrombin (factor II), methylenetetrahydrofolate reductase (MTHFR) and angiotensin 1-converting enzyme (ACE),

and wherein the presence of one or more <u>of the</u> mutations or polymorphisms indicates that the human subject has a genetic predisposition for <u>VT venous thrombosis</u>.

Claims 2 to 7. (Canceled)

Claim 8. (Currently Amended) The method of claim 1, wherein the method <u>further</u> provides a probability of developing VT of at least 98% in Caucasians, at least 85% in Asians, and at least 87% in Africans.

Claim 9. (Canceled)

Claim 10. (Currently Amended) The method of claim 1, wherein the at least eight VT-related molecules comprise nucleic acid molecules.

Claim 11. (Currently Amended) The method of claim 10, wherein the nucleic acid molecules are amplified from the <u>human</u> subject, thereby generating amplification products, and wherein the amplification products are hybridized with oligonucleotide probes that detect the one or more mutations or polymorphisms.

Claim 12. (Currently Amended) The method of claim 11, wherein hybridizing the oligonucleotides comprises:

incubating the amplification products with the oligonucleotide probes for a time sufficient to allow hybridization between the amplification products and oligonucleotide probes, thereby forming amplification products:oligonucleotide probe complexes; and

analyzing the amplification products:oligonucleotide probe complexes to determine if the amplification products comprise one or more mutations or polymorphisms in the VT-related nucleic acids, wherein the presence of one or more mutations or polymorphisms indicates that the <u>human</u> subject has a genetic predisposition for VT.

Claim 13. (Currently Amended) The method of claim 12, wherein analyzing the amplification products:oligonucleotide probe complexes comprises determining an amount of nucleic acid hybridization, and wherein a greater amount of hybridization to one or more of the mutated sequences, as compared to an amount of hybridization to a corresponding wild-type sequence, indicates that the human subject has a genetic predisposition for VT.

Claim 14. (Original) The method of claim 12, wherein analyzing the amplification products:oligonucleotide probe complexes includes detecting and quantifying the complexes.

Claim 15. (Original) The method of claim 11, wherein the oligonucleotide probes are present on an array substrate.

Claim 16. (Original) The method of claim 15, wherein the array further comprises oligonucleotide probes complementary to wild-type VT-related nucleic acid molecules.

Claim 17. (Original) The method of claim 16, wherein the wild-type VT-related nucleic acid molecules comprise oligonucleotide probes complementary to wild-type AT III, wild-type protein C, wild-type protein S, wild-type fibrinogen, wild-type factor V, wild-type factor II, wild-type MTHFR and wild-type ACE nucleic acid sequences.

Claim 18. (Currently Amended) The method of claim 1, wherein the at least eight VT-related molecules consist of sequences from AT III, protein C, protein S, fibrinogen, factor V, factor II, MTHFR and ACE.

Claim 19. (Currently Amended) The method of claim 1, wherein the <u>human</u> subject is in a group potentially at risk of developing a venous thrombosis.

Claim 20. (Currently Amended) The method of claim 19, wherein the <u>human</u> subject is pregnant, is in puerperium, is using oral contraceptives or hormone replacement therapy, has previous thrombosis history, has or will undergo prolonged immobilization, has a myeloproliferative disorder, has a malignancy, has or will undergo surgery, has a bone fracture, is of advanced age, has antiphospholipid antibodies, or combinations thereof.

Claim 21. (Currently Amended) The method of claim 11, wherein the nucleic acid molecules obtained from the human subject are obtained from serum.

Claim 22. (Currently Amended) A method of detecting genetic predisposition to <u>venous</u> thrombosis (VT) VT in a <u>human</u> subject, comprising:

applying amplification products to an array, wherein the array comprises oligonucleotide probes <u>each</u> capable of detecting <del>at least</del> the 143 mutations or polymorphisms listed in Table 1, and wherein the amplification products comprise nucleic acid sequences from AT-III, protein C, protein S, fibrinogen, factor V, factor II, MTHFR and ACE, obtained from the subject;

incubating the amplification products with the array for a time sufficient to allow hybridization between the amplification products and oligonucleotide probes, thereby forming amplification products:oligonucleotide probe complexes; and

analyzing the amplification products:oligonucleotide probe complexes to determine if the amplification products comprise one or more mutations or polymorphisms <u>listed in Table 1</u> in the AT-III, protein C, protein S, fibrinogen, factor V, factor II, MTHFR or ACE sequences, wherein the presence of one or more mutations or polymorphisms indicates that the <u>human</u> subject has a genetic predisposition for VT.

Claim 23. (Currently Amended) A method of selecting a venous thrombosis (VT) therapy, comprising:

detecting a genetic predisposition to VT mutation or polymorphism in at least one VT-related molecule of a subject, using the method of claim 1; and

if such at least one mutation or polymorphism is identified, selecting a treatment to avoid or reduce VT, or to delay the onset of VT.

Claim 24. (Currently Amended) The method of claim 23, further comprising administering the selected treatment to the human subject.

Claim 25. (Currently Amended) The method of claim 24, wherein the selected treatment comprises treating the <u>human</u> subject with an anticoagulant agent.

Claims 26 to 28. (Canceled)

Claim 29. (Currently Amended) A method of detecting a genetic predisposition to venous thrombosis (VT) in a human subject, comprising:

applying amplification products to the array of claim 13 to an array, wherein the amplification products comprise amplified nucleic acids obtained from the <u>human</u> subject, wherein the nucleic acids comprise coding or non-coding sequences from <u>antithrombin III (AT III) AT-III</u>, protein C, protein S, fibrinogen, factor V (FV), <u>prothrombin (factor II) factor II</u>, <u>methylenetetrahydrofolate reductase (MTHFR) MTHFR and or angiotensin 1-converting</u> enzyme (ACE), <del>ACE.</del>

incubating the amplification products with the array for a time sufficient to allow hybridization between the amplification products and oligonucleotide probes, thereby forming amplification products:oligonucleotide probe complexes; and

analyzing the amplification products:oligonucleotide probe complexes to determine if the amplification products comprise one or more mutations or polymorphisms in the AT III, protein C, protein S, fibrinogen, factor V, factor II, MTHFR, or ACE genes, wherein the presence of one or more mutations or polymorphisms indicates that the <u>human</u> subject has a genetic predisposition for VT.

Claims 30 to 35. (Canceled)